# HEMIPLEGIA IN CHILDHOOD.

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THE recent contributions of Strümpell (70, 71) on the subject of cerebral infantile paralysis have aroused a new interest in a symptom-complex which, although long recognized by foreign writers, has been much neglected by Americans and Englishmen. Although hemiplegia in childhood is often attended by certain peculiarities which entitle it to special consideration, none of the works on children's diseases, with the single exception of Money's (49) treatise which has just appeared, describes it or discusses the vexed question as to its pathology. Most of them speak of ordinary cerebral hemorrhage and embolism; but, with the exception of Money, none of them so much as mention the atrophy of the cortex so often met with, or give any full account of the special features of hemiplegia in childhood. This omission is the more singular, since many of the recent text-books on nervous diseases describe the affection with more or less fulness.

We owe to Cazauvieilh (14) the earliest description of that form of cerebral infantile paralysis which is now regarded as typical—the form due to atrophy of the convolutions—although he states that cases had been met with previously by his instructors in Paris. In 1853 Little (40), in England, in writing of "spastic rigidity of the limbs of the new-born" described cases of cerebral infantile paralysis, but he failed to distinguish these cases clearly from

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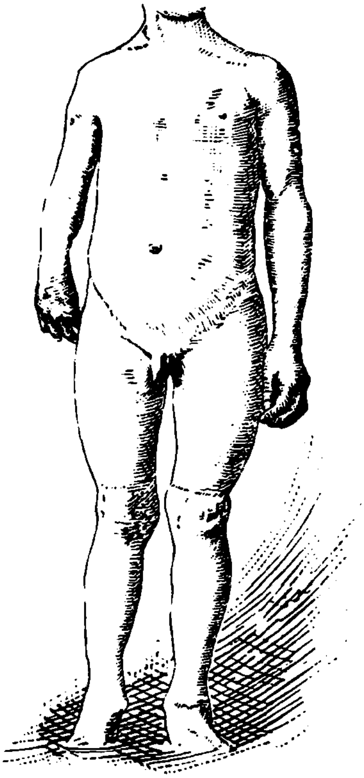
[1] Candidate's paper read by title at the meeting of the Am. Neurol. Assoc., July, 1887.

cases of spastic paraplegia from spinal disease, or to recognize the pathological changes underlying the affection.

During the past year, I have had the good fortune to see no less than ten cases of paralysis due to cerebral disease in childhood. None of these cases have as yet come to autopsy, so that I can add nothing to the study of the morbid processes involved, but clinical study certainly gives some hints as to a possible pathology in the cases which I shall report.

OBSERVATION I.—*Convulsions in infancy. Right hemiplegia with contracture. Convulsions, now often limited to right arm. Arrest of growth on paralyzed side.*[1]



Frederick K., 11 years old, the second child of German parents. His mother had weak, faint spells before he was born, and is said to have uterine disease. With this exception the family history is good. One child died of heart disease, five others are living and healthy. There is no history of syphilis or hereditary nervous taint. He was born at term, the labor being twelve hours in length and rather difficult, but instruments were not used. The head presented. After birth no abnormality was noted except blueness of the fingers. At the age of three months, he began to have convulsions, which gradually increased in frequency. When one year old, he began to get his teeth, dentition being difficult, and each tooth requiring to be lanced several times. The convulsions at this time grew worse, he had at times fifty or sixty a day, and sometimes ten or a dozen while his father was taking him to a doctor. Cutting the gums seemed to give relief. I could learn little as to the character of these convulsions except that they were general—he became rigid and all his limbs worked. Since dentition the convulsions have continued with more or less frequency. He has gone without any for two months at a time, and then he has had them quite frequently. March 26th, 1887, two days before he was brought to the hospital he had twelve, and the next day ten. The present convulsions begin with a feeling of something going down the right arm, which comes on long enough before the attack to enable him to go and lie down. In the severe attacks, everything looks dark to him; he can neither see nor speak; he cries out and works all over. Only in the severe attacks does he lose consciousness. Most of his attacks are confined to the right arm, and many of them to the right hand, and in these he is conscious and can speak. On May 23d, I saw him in a mild attack. There was no change of color in the face, and the right hand was the only part affected. He was perfectly conscious throughout and could answer questions. The pupils were not observed. The fingers of the right hand were strongly extended and abducted—a position which he could not put them into of his own will—and they twitched a little in adduction and flexion. The whole attack lasted forty or fifty seconds.

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[1] The illustrations are reproduced from photographs taken by Dr R. A. Kingman, of Boston.

The boy could not walk until he was three years old, and then the parents noticed that the right side was much weaker than the left, a condition which probably had come on some time before. He now walks with a limp, but the arm remains partly paralyzed, and there is a decided arrest of growth on that side. He could not talk until he was three years old, and now his speech is rather indistinct. The trouble is purely one of co-ordination, for he never uses the wrong word or is at a loss for a word; he reads and writes as well as could be expected, and his speech is always intelligent. In spite of his hemiplegia and convulsions, his general health is very good; he never has the headache, eats and sleeps well, and has no trouble of any sort. His parents say that his teacher considers him a very good scholar, and they regard him as very bright, as bright if not brighter than their other children. Two years ago, he was sent to Dr. Baker's home at Baldwinsville for his convulsions, but he grew very homesick and stayed but six months. Since that time he has taken bromide—how much or how constantly I could not learn—without much benefit. Borax—fifteen grains three times a day—was given, and since then the convulsions have been fewer and less severe. An attempt was made to control the individual attack by a ligature at the wrist, but it had no effect.

The boy is a little below the average height, but he is remarkably well developed, except on his paralyzed side, and well nourished. He appears very bright and quick-witted. The sense of smell is alike on the two sides. Vision is good; the eyes and pupils move naturally, and the field of vision and the fundus oculi are normal. The tongue is protruded straight. The right side of the face is smoother than the left, but he can draw his mouth better to the right; on whistling or laughing, the mouth is drawn a little more to the left than to the right. There is no difficulty in swallowing. The right side of the thorax is smaller than the left, but physical examination reveals nothing abnormal. The right arm is much atrophied, the hand is cold and cyanotic, and the arm is held against the chest, flexed at the elbow and wrist, strongly pronated, with the fingers flexed on the palm. The accompanying photograph, kindly taken for me by Dr. R. A. Kingman, of Boston, will show his ordinary attitude. Voluntary movements at the shoulder and elbow are possible, but he can neither supinate nor extend the wrist. The wrist can be flexed a little more, and there is a little movement of the fingers, chiefly in flexion. There is marked rigidity of the wrist on passive motion, and some of the elbow. The foot is in equino-varus, and the toes can be moved but little voluntarily; there is also marked resistance to passive motion at the ankle. The heel, however, reaches the floor on standing. There is no power of voluntary motion at the ankle, but motion at the hip and knee is fairly good. He walks fairly well, swinging his leg in the characteristic manner of a hemiplegic patient. The muscles of the arm reacted about alike on the two sides to a weak faradic current; except that on testing the extensors of the wrist and the supinators on the right the current had to be stronger, so that it was diffused, causing contracture of the antagonists, and the muscles tested did not respond as well. Sensation was everywhere normal. The epigastric, abdominal, and cremaster reflexes were normal; the plantar was normal on the left, but absent on the right. There was no triceps, radial, or ulnar reflex, or ankle clonus. The patellar reflex was exaggerated on the right, but very weak on the left, except by Jendrássik's method.

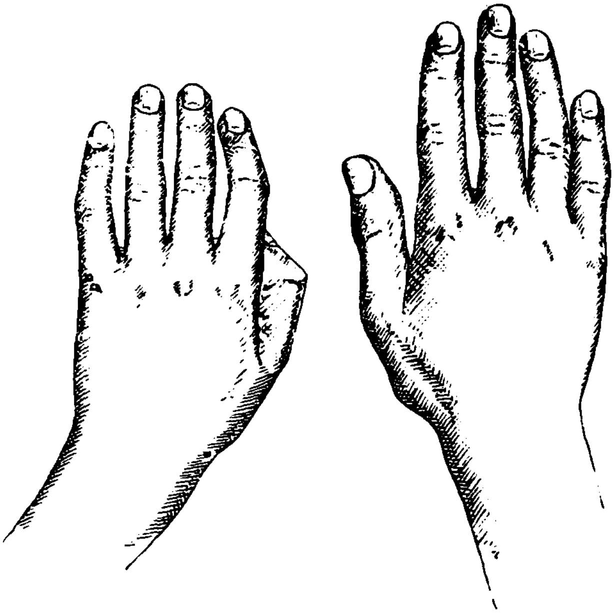
The following measurements were taken:

| | RIGHT. | LEFT. |
|---|---|---|
| Semicircumference of chest at nipples,. . . . . . | 11⅛ in. | 12⅛in. |
| Circumference of arm 4 inches above olecranon . . | 6 “ | 7½“ |
| “ “ “ 5 “ “ styloid process of radius | 6¼ “ | 7½“ |
| “ “ “ 2 “ “ “ “ “ “ | 4⅜ “ | 5⅜“ |
| “ “ hand at metacarpals, just above thumb, | 5½ “ | 6½“ |
| “ “ thigh 5 inches above upper edge of patella | 13½ “ | 14¼“ |
| “ “ calf in largest part, . . . . . | 9½ “ | 10 “ |
| Anterior superior spine to internal malleolus, . . . . | 23⅝ “ | 23⅞“ |
| Length of ulna, . . . . . . . . . . | 6½ “ | 7¼“ |

Clavicles about the same in length,
The right hand and fingers are decidedly smaller.

OBSERVATION II.—*Injury to head Convulsions. Paralysis of left arm and face without marked contracture. Arrest of growth in arm.*

James E., 14, born in Italy. He was in good health until he was six years and a half old when he fell from a window, striking his head. He was unconscious for three hours, and then made a complete recovery. Three months later he had a convulsion. Since then he has had convulsions at various intervals, averaging about one a week. It was not easy to get a clear account of these attacks from either the boy or his family. He is said to cry out, bite his tongue, and lose consciousness, but he does not fall. I could not learn whether the attacks were preceded by an aura or not. In former attacks, the face was drawn to the left and the left arm worked, but now neither the face nor the limbs are convulsed. Since having the convulsions, he has had loss of power in the left arm, which is now much smaller than the other. He has had no difficulty in walking, or paralysis of the leg, but lately he has complained that his legs ache, and that his left knee is sore. He has occasional frontal headache, but no vertigo. Lately he has had a little cough and palpitation and some pain in the chest. His appetite is good, but he has had a little diarrhœa for two or three months before coming to the hospital. He was given bromide of sodium, fifteen grains three times a day, which stopped his convulsions completely during the whole time that he was under observation—some five or six weeks.



The boy is well nourished and developed. The left arm is manifestly smaller than the right, and is cold and cyanotic. The eyes move naturally, the pupils respond to light, and the fundus oculi is normal. He can perform all movements with his left hand except adduction of the little finger, but all the movements are feeble. The dynamometer showed a grasp of 46 with the right hand and 22 with the left. There is no contracture or rigidity of tbe arm, but the fingers are slightly flexed and are a little stiff and cannot be completely extended. The photograph, taken like all the rest by Dr. Kingman, shows this slight contracture, and the smaller size of the hand. The boy was directed to hold both hands up against a screen as high as he could, and, as will be seen, he could not reach as far with the left. He cannot draw his face to the left, either voluntarily or on laughing. No cicatrix or depression of the skull could be detected. There is no paralysis or noticeable weakness of the legs, which are of the same size. The fingers of the left hand are smaller and smoother. The muscles of the arms react alike to a weak faradic current. The triceps and radial reflexes are slightly more marked on the left. The patellar reflex is absent, except by Jendrássik's method, when it is very weak. Sensation is normal. Examination of the chest revealed nothing.

The following measurements were noted :

| | RIGHT. | LEFT. |
|---|---|---|
| Circumference of arm 6 inches above olecranon . . . | 7¾ in. | 7 in. |
| " " " 3 " " " . . . . | 7¾ " | 7¼ " |
| " " " 5 " " styloid process of radius | 7¾ " | 6½ " |
| " " " 1 " " " " " " | 5⅞ " | 5⅛ " |

OBSERVATION III.—*Right hemiplegia with contracture. Some mental impairment. Absence of convulsions. Marked arrest of growth on paralyzed side.*

Wm. C., 11, of Irish parentage. He was born at term in one pain, the head presenting and the labor being natural and easy. When three weeks old he had scarlet fever mildly, and when five months old he had whooping cough. No history of any syphilitic or hereditary nervous taint could he obtained. He learned to walk at ten months, and after that walked and ran about like other children. About the same time he learned to talk. When a year and ten months old he was put to bed in his usual health, and nothing was heard from him during the night. The next morning it was found that he could not walk or stand alone, and later in the day his mouth was drawn to the left. Since that time the whole right side has been paralyzed, but there has never been any disturbance of speech. For three months he was in bed, but after that he got up and began to get about. At present he goes to school, and plays with the other boys, and in spite of his paralysis, can get over the ground pretty fast. Except for an attack of measles some years ago his health has been good, although he is not very vigorous. He began to go to school at the age of five, but thus far he has not distinguished himself. He is still in the primary school, and spent four years in one room. His teacher calls him a good boy but a poor scholar. He can read some and write a little with his left hand. His father states that his memory is very poor. Until he was seven years of age his mother says that both sides of his body grew alike, but after that his right side did not grow as fast, so that it is now much smaller than the other. He has never had convulsions or *petit mal*, and is not subject to headache. He can talk perfectly well. There are no thoracic, abdominal, or urinary symptoms.

The boy is of the average size, rather poorly nourished, and presents a pronounced arrest of development on the right side, even the face seeming smaller on that side. He answers questions readily and comprehends what is said to him, but he is rather stupid. He is said to be able to read his school-books quite well, but on trying him with the printed diet list of the hospital he read the short words well enough, but he had much difficulty with the longer and more unusual words. The pupils and eyes moved naturally, the field of vision was good, and the fundus oculi was normal. The tongue was protruded straight. He did not wrinkle the right side of his forehead as much as he did the left in raising his eye-brows, but he closed his eyes naturally. He could draw his face to the right fairly well, but on laughing it was drawn to the left, and in repose the left naso-labial fold was more marked, and the left angle of the mouth was a little higher. This paralysis on mimetic but not on voluntary movement, seen here and in Obs. I., is of interest as an indication of the existence of separate channels for innervation. Rosenbach,[1] who has recently called attention to the subject, claims that the movements of expression are innervated through the optic thalami. The apparent paresis of the upper branch of the facial is remarkable in intracerebral disease. The accompanying photograph will show the atrophy and deformity. His right arm, which is much atrophied, is held to the side, flexed at the elbow and wrist, strongly pronated, and the fingers semi-flexed. The skin is cold and cyanotic. There is great resistance on attempts at passive motion, owing to the great muscular contracture; at the wrist this cannot be overcome by any safe expenditure of force. He can move the shoulder and elbow a little, but the only movement possible of the hand is further flexion of the fingers. The thigh is flexed somewhat on the pelvis and rotated inwards, and the leg is flexed on the thigh. The gastrocnemius is greatly contracted, drawing the heel from the ground, so that he walks on the ball and inner edge of his foot—the foot being greatly deformed, and in a position of marked equino-varus. This deformity cannot be overcome by any attempts at replacement. There is fairly good movement of the hip, some of the knee, but none of the foot or ankle. The pelvis is tilted to the left to counteract the deformity. There is a compensating lateral curvature of the spine which makes the thorax seem smaller on the paralyzed side, although it is not. He gets over the ground pretty fast, using the right leg as an occasional point of support, and hopping much of the way on the left foot. The muscles of the arm reacted alike on the two sides to a weak faradic current. Sensation was normal. Examination of the chest was negative. The abdominal and cremaster reflexes were normal. The plantar was normal on the left but absent on the right. The biceps, radial, and patellar reflexes were present, and were slightly more

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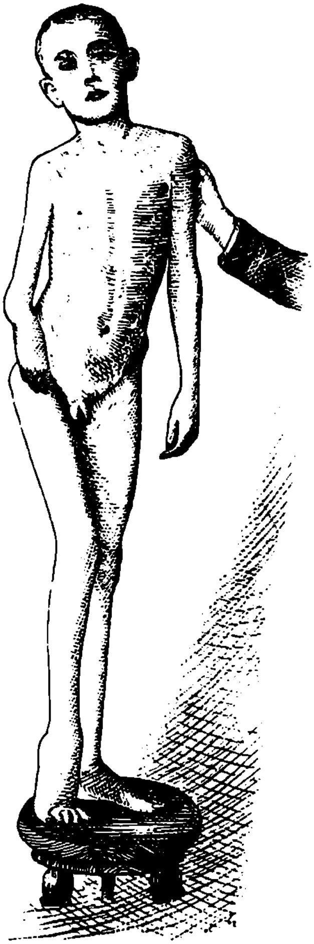
[1] P. Rosenbach, "Zur Lehre von der Innervation der Ausdrucksbewegungen." Neurol. Centralblatt, June 1st, 1886.



marked on the right side. The was no wrist, patellar, or ankle clonus.

The following measurements will show the degree of wasting on the paralyzed side:

| | RIGHT. | LEFT. |
|---|---|---|
| Semi-circumference of chest at nipples, . . . . | 11½ in. | 11½ in. |
| Circumference of arm 4 inches above olecranon, . . | 5½ " | 6½ " |
| " " " 3 " " styloid process of ulna, | 4½ " | 6¼ " |
| " " " 1 " " " " " " | 3⅞ " | 4⅞ " |
| " " thigh, 5 " " upper border of patella, | 10¼ " | 11¾ " |
| " " calf in largest part, . . . . | 7¼ " | 9⅜ " |

| | RIGHT. | LEFT. |
|---|---|---|
| Anterior superior spine of ileum to internal malleolus, . | 28¼ in. | 29 in. |
| Length of ulna, . . . . . . . . . | 6½ " | 7⅝ " |

Fingers of right hand and hand itself much smaller.

OBSERVATION IV.—*Right hemiplegia of gradual onset. Temporary aphasia. Slight arrest of growth. No rigidity, contracture, or convulsions.*

Kate H., 12 years old, of Irish parentage. The family history shows no syphilitic or hereditary nervous taint. Eleven years ago she had diphtheria, three years ago whooping cough, and two years ago measles. Except for these illnesses she has been well until a year ago. At that time, on her return from school, where her work had not been especially hard, she had a "reeling in her head," and fell over on the floor. She was picked up and probably had some difficulty in walking immediately after. There was no loss of consciousness, but her right side gradually became paralyzed, and her face was drawn to the left. For six weeks she was confined to her bed, then she gradually got able to move about and to use her limbs, and since then she has steadily but slowly improved. She never had convulsions, but during her confinement to the bed she complained greatly of headache and heat in the head, and ice was applied to the head by her physician's orders. Six months ago she had some twitching of the right hand, which has disappeared. For three months she could not talk so as to be understood, but now she talks without any noticeable defect. During the first of her illness she was rather slow and dull in her mental processes, but now her mental condition is natural. At present she has some headache, chiefly frontal or in the left temporal region, and a little vertigo. There never have been any thoracic symptoms, and before this attack she could run and play like other children. The digestive functions are performed normally. She sleeps well, and there has been no change of disposition.

The pupils and eyes move naturally; the field of vision and the fundus oculi are normal. She cannot draw her mouth to the right, but there is no inequality on smiling. She can move her arm and leg in all directions, but without much strength. Dorsal extension of the right foot is not performed as well as that of the left, and extension and abduction of the fingers of the right hand is attended with a little tremor. There is no contracture or rigidity, but the limbs are a little smaller on the right. She always was right-handed, but since her illness she has learned to write with the left hand. The heart and lungs were normal on examination. The plantar reflex is diminished on the paralyzed side; the abdominal and epigastric reflexes are absent on both sides. The triceps reflex was alike on the two sides; the radial and ulnar were absent, the patellar was exaggerated, and rather more marked on the right side. There was no clonus or front tap contraction. Sensation was perfectly good. The muscles of the face and arms react alike on the two sides to a weak faradic current. Dr. W. N. Bullard, who saw her previously at the Boston Dispensary, and kindly permitted me to make use of his notes, reports that the galvanic reactions were tested by Dr. J. A. Jeffries, who found that the extensor communis digitorum showed KaSZ > AnSZ with $\frac{3}{10}$ Ma.; the right peronei muscles, KaSZ > AnSZ $\frac{6}{10}$ Ma.; the rectus femoris, KaSZ=AnSZ; everywhere else KaSZ > KaOZ and AnSZ > AnOZ. The following measurements show the difference in size.

| | RIGHT. | LEFT. |
|---|---|---|
| Circumference of arm, 5 inches above olecranon, . . . | 6$\frac{1}{2}$ in. | 7$\frac{1}{4}$ in. |
| " " " 5 " " styloid process of radius, | 6$\frac{3}{8}$ " | 6$\frac{5}{8}$ " |
| " " " 1 " " " " " " | 4$\frac{5}{8}$ " | 4$\frac{7}{8}$ " |
| " " thigh, 5 " " upper border of patella, | 12$\frac{1}{2}$ " | 12$\frac{1}{2}$ " |
| " " calf in largest part, . . . . | 10$\frac{1}{8}$ " | 10$\frac{3}{8}$ " |

The right ulna was as long as the left, the semicircumferences of the chest were the same, and the legs were of the same length. As the paralysis is not of long duration, and as the child is already pretty well grown, we should not expect much of an arrest of growth in the bones as yet, if at all.

OBSERVATION V.—*Cephalalgia, probably of syphilitic origin. Right hemiplegia. Arrest of growth on right side. No contracture, but exaggeration of tendon reflexes.*

Michael R., 21, S., Plumber. Came to the out-patient department of the Boston City Hospital complaining of severe headache, weakness, and malaise. He seemed so ill that no careful examination was made. He stated then that his headache had lasted for five or six weeks, that he had used alcohol to excess, and that he had never had syphilis. For a month or six weeks he had noticed that his face was drawn a little to the left, and that his right side was weaker than thele ft. On stripping him there was found to be a distinct right hemiparesis, and a marked difference in the size of the limbs on the two sides of the body. Dr. Gleason, who was acting as externe, reported that the muscles of the arms reacted alike on the two sides to a mild faradic current. I am obliged to quote my own observations from memory, but I am indebted to Dr. C. F. Folsom, to whose service he was admitted, for kindly furnishing me a copy of the records of his case while in the hospital, as follows:

"Admitted June 15th, 1887. Family history good. Left-sided pleurisy two years ago. No other sickness. No syphilis. Drinks to excess. Not feeling well for two weeks, general weakness, frontal headache, loss of appetite. Bowels constipated. No abdominal or febrile symptoms. Last two days vomited three times. About six months ago noticed right hand smaller than left. No pain in right arm or leg, but they are not as strong as formerly; finds it difficult to use right hand in fine work. Eyesight and eyes all right.

"Well-developed and nourished. Tongue moist, with a white coat, protruded a little to the right. Pupils normal. Left corner of mouth drawn up a little. More contraction of left muscles than right when he knits his brows. Right grasp weaker than left. Heart's apex in or just outside of mammillary line. Prolonged first sound at apex; action a little uneven. Lungs and abdomen negative. Both knee-jerks exaggerated, especially right. Considerable tremor of leg when lightly tapped. Ankle clonus. Wrist clonus a little more marked right than left. On walking, right leg seems stiff, and foot occasionally drags. Stands well with feet together and eyes shut. No tenderness of shins. No neuritis or œdema. Patient right-handed. Left upper arm 2.5 cm. larger than right; forearm 1 cm. larger; hand at the metacarpo-phalangeal joints 2 cm. larger; right arm to end of middle finger 1 cm. shorter.

"June 23d.—Has had two or three attacks of extremely severe headache, relieved by morphine, and diminished by iodide of potassium, gr. xl., t. i. d. Now slight headache. Some dizziness and nervousness. Facial paralysis seems to be increasing. Up and about daily.

"June 28th.—Discharged at own request, against advice."

In spite of the patient's statement, it did not seem possible either to Dr. Folsom or myself that any recent lesion could cause such an atrophy of the right side, involving the bones; and furthermore, the benefit derived from iodide made us strongly suspect some syphilitic taint. It seemed to us not unlikely that he had had a hemiplegia in childhood, from which he had recovered, but that it had caused an arrest of growth on that side, and possibly an exaggeration of the tendon reflexes. This had passed unnoticed until recently, when a syphilitic process (endarteritis?) had developed at the site of the old lesion, as it is prone to do, causing weakness of the right side, and perhaps facial paralysis, and, with its attendant symptoms of headache and malaise, had called his attention to his right side, which he discovered for the first time to be smaller than the left. It is a matter of common experience that symptoms that do not cause discomfort are often overlooked, and, in a patient whose mental powers were as untrained as this man's, such a hypothesis is by no means improbable.

A glance at the illustrations to this article—notably those of Observations I. and III.—or at the patients themselves, would lead one very naturally to make a diagnosis of an old anterior poliomyelitis; but more careful examination will show that in all these cases the lesion is without doubt cerebral. The hemiplegic distribution of the paralysis, the implication of the facial muscles, the exaggerated reflexes, the spastic contracture seen chiefly in the arm, the normal electrical reactions, the presence of convulsions, all point to a lesion in the brain and not in the anterior cornua of the cord. It is a cerebral infantile paralysis and not a spinal. In spinal paralysis hemiplegia is rare and the face is very rarely if ever involved; the tendon reflexes are absent or at least diminished, convulsions, except in the initial stage, are never present, and the characteristic reaction of degeneration, or the absence of faradic reaction or of both faradic and galvanic reactions, is to be expected.

There is one point of distinction in the cases reported which seems to me of decided importance, and that is the existence of epileptiform attacks after any symptoms of active disease have passed away. This I believe to be of great value in diagnosis as a means of differentiation between a lesion involving the cortex and a lesion farther down in the brain, deep down in the centrum ovale or in the region of the basal ganglia. In the first two cases I believe that the lesion is cortical; in the third and fourth that it is central; the fifth I imagine is central, but the impossibility of getting any definite history from the patient renders it useless to attempt to draw any conclusions from it, and I shall therefore leave it out of account.

Considering first the cases where epileptiform attacks were absent (Obs. III. and IV.), in Obs. III. the lesion has destroyed the left motor tract somewhere between the cortex and the pons, probably in the internal capsule, and has been followed by a secondary degeneration of the right crossed pyramidal tract in the cord. In Obs. IV. there is little or no secondary degeneration, and the lesion is probably not one which destroys the tract, but one which causes a break in the nerve conduction by pressure upon it, and it very likely lies in the lenticular nucleus or the corpus striatum.

The pathology of these cases is not without interest, although it has been less discussed, owing to the much greater interest aroused of late in the study of the pathology of cortical lesions in children, of which I shall speak later. The lesions in or about the basal ganglia giving rise to hemiplegia are the same as those in adults—abscess, tumor, embolism, thrombosis, and hemorrhage. That in

Obs. III. or IV. there is any progressive trouble, such as abscess or tumor, seems extremely improbable from the course of the cases, and the absence of any special symptoms, such as headache, vertigo, or optic neuritis.

At the last meeting of the American Neurological Association Dr. Sachs (61), in a paper of which only an abstract has yet appeared, called attention to the occurrence of intracerebral hemorrhage in the young, and laid especial stress on the absence of epileptiform attacks as a diagnostic point between intracerebral and meningeal hemorrhage. That intracerebral hemorrhage does occur in children has been repeatedly proven by autopsy, although most writers on children's diseases claim that it is extremely rare. Dr. Sachs stated that, according to certain observations made by Osler, miliary aneurisms occur in children; and furthermore, that Recklinghausen claims that fatty degeneration of the cerebral arteries, permitting transudation of blood, was not infrequent. In the discussion that followed Dr. Sachs' paper, Dr. Zenner called attention to the fact that there may be an embolism from endocarditis when no adventitious murmur is to be heard in the heart. The diagnosis between embolism and hemorrhage is so difficult as often to be impossible, and although in these two cases I am inclined to believe that there has been a hemorrhage, I do not feel that such a diagnosis could be confirmed without an autopsy.

Returning now to Obs. I. and II., in which epileptiform attacks were present, and in which the lesion was thought to be cortical, we enter upon a question in pathology which of late has been widely discussed. The discussion of the character of the lesion in Obs. II. need not detain us long. Evidently of traumatic origin, there has probably been a hemorrhage, either in the meninges or involving the cortex itself, yet not severe or extensive enough to destroy its functions. The absence of severe headache, vertigo, and other general symptoms makes the diagnosis of any other morbid process, such as meningitis or tumor less probable.

In Obs. I., however, the case is different. Here we have a morbid process coming on spontaneously, involving the cortical gray matter of the left central convolutions, most markedly in the centre for the hand, but involving also the centres for the face, arm, and leg, and the motor centre for speech. From the cortex there has been a descending degeneration of the fibres of the pyramidal tract down into the cord. This is one of the cases to which Strümpell (70, 71) has given the name of acute poliencephalitis, regarding them as similar in respect to their morbid processes to the cases of acute anterior poliomyelitis.

The disease attacks children in the early years of life, especially under six years of age, and comes on either during a period of perfect health or especially after some acute disease, particularly the exanthemata. It may begin with fever, delirium, vomiting, and diarrhœa, or, very commonly, with convulsions, and Strümpell suggests that poliencephalitis may be the cause of death in those cases where a child dies suddenly with acute cerebral symptoms, such as convulsions. On recovering from this initial stage the child is found to be paralyzed, usually hemiplegia, the arm, leg, and lower part of the face being generally affected, although strabismus is sometimes seen. The arm is generally more helpless than the leg, as in the hemiplegia of adults. Contracture develops as in adults, and there is also a pronounced arrest of growth in the paralyzed limbs, involving the bones as well as the muscles. There may be a slight quantitative diminution of electrical excitability in the paralyzed muscles, although this is rare, but there are never any quantitative changes. Sensation is normal. The tendon reflexes are exaggerated on the paralyzed side and sometimes on both sides; but the cutaneous reflexes are absent or diminished. In right hemiplegia there may be disturbances of speech. Epileptiform attacks generally persist, and in many cases there are other motor disturbances—chorea, athetosis, ataxia, or associated movements. The child is very apt to be imbecile or idiotic. In the cases that have come to autopsy, porencephalia—a loss of substance or atrophy of the convolutions—has been observed, and many observers have found traces of old inflammatory disturbance, spider and fatty granular cells. Strümpell, moreover, is inclined to regard this disease, as well as acute anterior poliomyelitis, as of infectious origin—a hypothesis which has received a remarkable support from a case cited by Möbius (48). Two children in the same family were attacked within twenty-four hours of each other with fever, loss of appetite, and restlessness. One child, a girl, in a day or two developed a typical anterior poliomyelitis, and her brother an equally typical spastic hemiplegia, with post-paralytic chorea.

Strümpell (70) closes his paper by saying that we must bear in mind that the hemiplegia of children may be due to other causes of which I have already spoken. Bernhardt (5) adds that if, "either suddenly while in apparently perfect health, or in the beginning, or during the course of convalescence from an acute febrile disease (especially the exanthemata), we notice in children in the first years of life a sudden outburst of unilateral convulsions, lasting for hours and followed by hemiplegia with the peculiar symptoms described (contractures, hemichorea, hemiathetosis, associated movements, etc.); and if, beside these symptoms, the psychical anomalies and especially epilepsy persist, we are justified, in my opinion, in recognizing this special form of cerebral infantile paralysis, this spastic cerebral infantile hemiplegia, as a more or less distinct *clinical* type."

So far most observers substantially agree. They also admit that the lesion of this "distinct clinical type" is situated in the motor region of the cortex, but the nature of the morbid process is still a matter of dispute. In the majority of cases the result of the process is porencephalia—a loss of substance in the brain involving the convolutions, or a cicatricial thickening and atrophy of the cortex. Whatever the primary process may be, it leads ultimately to this. Dr. Sarah J. McNutt (44), in a paper read before the American Neurological Association three years ago, tabulated thirty-four autopsies, of which thirty-two showed wasting or porencephalic defects in the cortex. I have collected twelve other cases, in nine of which a similar condition was found [Bernhardt (5), Binswanger (7), Jendrássik and Marie [two] (33), Kast [two] (36), Mordet [50], Sander (62), Warner and Beach (73)]. One case [Seeligmüller (63)] revealed meningitis with softening of the cortex; in another [Beach (3)], the parietal convolutions were firmer and "coarser," *i. e.*, less well developed, and the microscope showed a great increase in the number of vessels, with distention of the vessels, and extensive infiltration with leucocytes, especially in the perivascular spaces. In the last case [Fraser (21)] there was a hypertrophy of the parietal lobe on the side opposite the paralysis, but the microscope revealed a defective development of cells.

The cause of this change in the cortex is still uncertain. In some cases where the trouble was congenital, Cazauvieilh (14) claimed that there was a defective development of the brain due to some intra-uterine disturbance. Little (41) attributed the trouble in his cases to injury of the brain received at birth. Strümpell's theory of an inflammatory process involving the cells of the gray matter of the cortex has already been referred to, and it is supported by Eichhorst (18). Kundrat 37) believes that porencephalia may be either congenital—a defect of development—or acquired. The acquired form follows the distribution of the arteries and is often of vascular origin, arising from an anæmia without arterial lesion, depending upon vascular weakness. This anæmia may be consecutive to pressure during labor. Porencephalia, he states, is a loss of substance from some destructive process in the brain, arising from hemorrhage, thrombosis, embolism, or anæmia without arterial lesion. Wood (76) and Bastian (2) assert that spastic infantile hemiplegia is due to cortical hemorrhage, but they do not say whether the hemorrhage may give rise to porencephalia; Ross (59), however, claims that it does. Jendrássik and Marie (33), however, could find no traces of hemorrhage or softening as a starting point for the sclerosis. In their two cases they found the perivascular lymph spaces greatly dilated, containing spider and fatty granular cells. This they regard as the primary process with a secondary diffuse process leading to sclerosis and atrophy following it, the exact nature of the change being still in doubt. Henoch (29) finds an interstitial growth of the neuroglia—a sclerotic process—with fatty granular cells and corpora amylacea. In this connection it may be said that Jastrowitz (32) does not regard the mere presence of fatty granular cells as proof of an inflammatory process, but only their aggregation into foci. The development of the medullary sheaths in the fœtal brain is normally attended with the formation of fatty granular cells. Steffen (69) attributes the trouble, perhaps, to a meningitis. Gowers (24) maintains that these cases of hemiplegia are due to a venous thrombosis. This view is corroborated by a case reported by Heubner (30), where, in addition to a genuine porencephalia, a thrombus was found in the right artery of the fissure of Sylvius. The patient also had endocarditis. Lambl (38), who examined a case of congenital brain defect, gives as the causes of porencephalia congenital defect, aplasia from syphilitic endarteritis or embolism, loss of blood-supply, interstitial encephalitis, a fatty degenerative process, Kundrat's anæmia without arterial lesion, and perhaps hydrocephalus.

The latest contribution to our knowledge of the pathology of the affection has been made by Kast (36), who had an opportunity to examine two cases at an earlier stage in the disease than any that have been reported. He agrees with Bernhardt in thinking the lesion is not a pure poliencephalitis, but also a chronic leuko-encephalitis, a diffuse sclerosis of the gray and white matter of the cerebrum. He failed to see the initial stage of perivascular changes described by Jendrássik and Marie, although he considered it impossible to decide from his data whether vascular changes were or were not the primary trouble; nor did he find Gowers' thrombosis of the veins. He believes it impossible at present to decide as to the primary change, owing to the lack of examinations of cases in the initial stages.

At present, therefore, it seems safe say that probably several processes may give rise to porencephalia—hemorrhage, embolism or thrombosis, congenital defect or acute encephalitis, which may be analogous to acute anterior poliomyelitis and may possibly be infectious.

The prognosis must always be guarded. In Obs. I. and II., I have thus far been fortunate in holding the convulsions in check, but they often persist. Mild cases of paralysis, as in Obs. IV. and V. may make a tolerable recovery by the aid of tonics, cod-liver oil, massage, and electricity; but when there is pronounced secondary degeneration, as in Obs. I. and III., the prospect of recovery is of course hopeless. The patient may live to a fair age, although Henoch (29) claims that few outlive their twentieth year, but it must be remembered that they never have a sound brain, and thus must fall behind in life's race. Idiocy and imbecility are common sequelæ, although not present in the cases reported, and Mercklin (46) thinks that in those whose minds are not at first affected there is a lessened resistance and an increased vulnerability of the brain, so that later in life various psychoses may develop. Persistent and patient work, however, in the way of massage and gymnastics, may produce some gain even in bad cases.

Concerning two of the symptoms, I wish to speak further. Although four of these five cases were cases of right hemiplegia, the disturbances of speech were insignificant. In some cases aphasia may persist, so that the patient may remain mute for life, although Bernhardt (5) questions whether congenital aphasia, unattended with idiocy or deafness, ever exists. Clarus (17) believes that where there has been a destruction of the speech centres the prognosis is bad, but with slight and transitory disturbances of them it is good. Bernhardt thinks that aphasia in children is generally temporary, and usually of the motor type. In children, the right hemisphere may become educated as a speech centre, and thus acquire the power of speech after the centres in the left hemisphere have been destroyed. In support of this theory, he cites an observation by Kahler (35), where a woman had right hemiplegia from congenital atrophy of the convolutions, including the left third frontal, with perfect power of speech. At the age of thirty-five she had apoplexy, causing left hemiplegia, from which she died. After her attack she was completely aphasic. At the autopsy a fresh lesion was found in the right hemisphere.

The other symptom to which I wish to call attention is the very marked arrest of growth in the paralyzed limbs. Seeligmüller (63) and Henoch (29) regard it as merely an atrophy from disuse, and assert that it is never so great as in anterior poliomyelitis. Such an assertion is untenable, for in some of these cases the atrophy is as pronounced as in poliomyelitis, and, as Förster (20) says, no atrophy from disuse could affect the growth of the bones. In certain cases of old hemiplegia in adults, Charcot,[1] Pitres,[2] and Brissaud[3] have found that, when there had been atrophy of the paralyzed limbs, the secondary degeneration had extended from the lateral columns to the motor cells in the anterior cornua, which they found atrophied. In some of these cases, however (Obs. II. and IV.), the signs of secondary degeneration in the lateral tract are very slight. In these cases, as in those of Ranke (54) and Seeligmüller (63), the electrical reactions were normal; Steffen (69), however, has noted a quantitative diminution, and Henoch (29) says that when the muscle is entirely wasted they disappear. I have not found this diminution in the hemiplegia of childhood, but I have seen it in adults, and in one case I have seen AnSZ > KaSZ which is, as far as my knowledge goes, unique.

In the cases reported, the various disturbances of motion—chorea, athetosis, ataxia, and associated movements—so often noted by other observers, were absent, except, perhaps, in the earlier course of Obs. IV. In the other cases of cerebral infantile paralysis which I have seen, however, such disturbances held a prominent place in the picture of the disease; and at some future time I shall hope to present a study of them.

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[1] Charcot, "Leçons sur les maladies du système nerveux." I., 55, Paris, 1872.

[2] Pitres, "Note sur un cas d'atrophie musculaire consécutive à une sclérose laterale secondaire de la moelle épinière." Arch. de physiol. norm. et path., p. 567, 1876.

[3] Brissaud, "De l'atrophie musculaire dans l'hémiplegie." Rev. mensuelle de méd. et de chir., p. 618, 1879.

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[1] See also this Journal and number, p. 503, 1887.